# Periodontal Disease: Diagnosis and Management

**Elaine Embrack Ridgeway, RN, MSN, CFNP**


## ABSTRACT

Approximately 50% of all adults in the United States have some form of gingivitis, and 80% have experienced some degree of periodontitis. Despite the use of fluoride, a critical element in the prevention of periodontal disease, many children and adults still suffer needlessly from this "silent" destroyer of the gum tissue and its underlying bone structures. This article highlights the prevalence, pathophysiology, classifications, health risk factors and clinical management of clients with periodontal diseases. Regardless of the health care setting, the advanced practice nurse plays a critical role in the prevention, early diagnosis and intervention of periodontal disease.


## INTRODUCTION

Gingival and periodontal diseases, in their various forms, have afflicted humans since the dawn of history. For centuries, the prevalence of periodontal diseases have continued to escalate as a major public health problem (Carranza, & Newman, 1996). Despite the use of fluoride, a critical element in the prevention of periodontal disease, many children and adults still suffer needlessly from this "silent" destroyer of the gum tissue and its underlying bone structures (www.cdcinfo@cdc.gov).

Approximately, 50% of all adults in the United States have some form of gingivitis, and 80% have experienced some degree of periodontitis. The average adult over age 40 years has 10 to 17 decayed, missing or filled permanent teeth. It is estimated that 22% of adults 45 years of age and 50% of adults 65 years of age and older are edentulous. Periodontal disease is responsible for more than 40% of all tooth extractions and is the major cause of tooth loss in individuals over age 45. Ninety-five percent of elderly persons have chronic periodonitis, with more than one-third experiencing moderate to severe forms of periodontal disease with at least one site of tooth attachment loss of 6 mm or greater (www.cdcinfo@cdc.gov).

This article highlights the prevalence, pathophysiology, classifications, health risk factors and clinical management of clients with periodontal diseases. Regardless of the health care setting, the advanced practice nurse plays a critical role in the prevention, early diagnosis and intervention of periodontal disease. While examining the oral cavity, it is imperative that the clinician be alert for obvious symptoms of oral disease and institute effective clinical management, including counseling, education and follow-up care.

## PATHOPHYSIOLOGY

Periodontal diseases involve the inflammation and degeneration of tissues that surround and support the teeth. This includes the gingiva, alveolar bone, periodontal ligaments and cementum. Periodontal diseases are generally chronic in nature and can persist in the absence of treatment. It most commonly begins as gingivitis and progresses to periodontitis as a result of exposure of the periodontium to dental plaques and biofilms that accumulate on the teeth to form bacterial masses at or below the gingival margin (American Academy of Periodontology, 1998). Dental plaques are complex, generally containing more than 400 species of bacteria. These bacterial organisms release toxic products into subgingival sites that stimulate a local inflammatory response and results in gingival ulceration around the teeth (Powell, 1998). When gingival ulcerations occur, bacteria and their chemical byproducts, such as lipopolysaccharides, peptidoglycan fragments and hydrolytic enzymes often enter into systemic circulation and result in a systemic inflammatory response (Powell, 1998).

## CLASSIFICATIONS

Periodontitis (inflammation of the gingiva) is classified according to the rate of progression (slow or rapid) and age of onset (adult periodontitis or early onset or juvenile periodon-

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**Table 1.**
MAJOR CAUSATIVE FACTORS IN THE DEVELOPMENT OF PERIODONTITIS AND ADDITIONAL ORAL PATHOLOGY.

| Causative Factors | Additional Oral Pathology Manifested with Periodontitis |
|---|---|
| ***Nutritional Deficiencies*** | |
| ◆ Thiamine (Vitamin B1) Deficiency (Beriberi) | Hypersensitivity of oral mucosa; minute vesicles (simulating herpes) on the buccal mucosa under the tongue or on the palate; erosion of the oral mucosa |
| ◆ Riboflavin Deficiency (Hyporiboflavinosis) | Glossitis; angular cheilitis |
| ◆ Niacin Deficiency (Pellagra) | Glossitis; gingivitis; generalized stomatitis; acute necrotizing ulcerative gingivitis |
| ◆ Folic Acid Deficiency with Sprue | Generalized stomatitis; ulcerative glossitis; cheilitis |
| ◆ Vitamin C (Ascorbic Acid Deficiency) Deficiency (Scurvy) | Hemorrhagic, edematous gingivae; loosened teeth due to diminished bone formation |
| ***Endocrine Disorders*** | |
| ◆ Hyperpituitarism | Marked overgrowth of the alveolar process that causes an increase in the size of the dental arch and increased spacing of the teeth. |
| ◆ Hypopituitarism | Decreased growth in the dental arch with crowding and malposition of the teeth |
| ◆ Diabetes Mellitus | Type I (uncontrolled): cheilosis with a tendency toward drying and cracking; burning sensation of the oral mucosa; xerostomia (dryness of the mouth due to decreased salivation); alterations in the oral flora with greater predominance of Candida albicans, hemolytic streptococci and staphylococci; diabetic periodontoclasia (degenerative changes); enlarged gingiva; sessile or pedunculated gingival polyps (poorly attached to gums); loosened teeth |
| ***Hematologic Disorders*** | |
| ◆ Leukemia | Bacterial gingivitis |
| ◆ Anemia | Gingival pallor |
| ◆ Iron Deficiency Anemia | Glossitis; ulcerations in the oral mucosa |
| ◆ Sickle Cell Disease | Glossitis |
| ◆ Thrombocytopenia Purpura | Spontaneous bleeding; gingival edema and friability |
| ◆ Immunologic Disorders | Gingivitis with oral ulcers |
| ***Cardiovascular Disorders*** | |
| ◆ Arteriosclerosis | Gingivitis associated with circulatory impairment |
| ◆ Congestive Heart Failure | Severe marginal gingivitis with periodontal destruction |
| ***Toxic Exposures*** | |
| ◆ Bismuth Intoxication | Ulcerative gingivostomatitis; bluish-black discoloration of the gingiva |
| ◆ Lead Intoxication | Change in gingival pigmentation; marginal gingival ulceration |
| ◆ Mercury Intoxication | Gingival ulceration |
| ◆ Benzene Exposure | Gingival bleeding |
| ***Psychosomatic Disorders*** | Grinding or clenching teeth<br>Excessive nibbling or chewing on foreign objects (i.e., pencils, pipes, etc.)<br>Nail biting<br>Excessive tobacco use<br>Betel nut chewing |

Source: *Periodontal Disease: Diagnosis and Management*

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titis). Other forms include necrotizing ulcerative periodonitis and refractory periodontitis (Carranza, & Newman, 1996).

Slowly progressive periodontitis (SPP) involves chronic inflammation of the gingiva with pocket formation and bone loss. The signs and symptoms include increased tooth mobility and pathologic migration of the inflammatory process. Although SPP can be preceded by long-standing chronic gingivitis, the destructive features are usually seen at age 35 years or older and usually correlated with the amount of plaque build-up on teeth (Carranza, & Newman, 1996).

Slowly progressive periodontitis may also develop secondary to systemic diseases, such as neutropenias, leukemias, AIDS, diabetes mellitus, Crohn's disease, Addison's disease and thyroid disorders (*Merck Manual*, 1998). Drug-induced SPP is associated with the use of oral contraceptives, cancer chemotherapy, phenytoin, cyclosporines and calcium channel blockers, drugs that often cause fibrotic gingival hyperplasia. Hypovitaminosis, including scurvy and pellagra, can also cause SPP, which will be manifested by inflamed, hyperplastic, engorged and hemorrhagic gingiva. In patients with scurvy, a vitamin C deficiency, the gingiva may appear ecchymotic with periosteal destruction and loose teeth. Pellagra, a deficiency in nicotinic acid, causes the gingiva to become inflamed, hemorrhagic and prone to secondary infections; the lips may appear red and cracked, while the tongue becomes smooth and bright red (Carranza, & Newman, 1996).

Rapid progressive periodontitis (RPP) can occur at an early age (before the end of puberty) or during the adult years. The signs and symptoms are usually associated with a lack of clinical inflammation or marked inflammatory changes; often there is a scant amount of plaque and calculus. As the disease progresses, deep pockets form in the gingiva, along with rapid bone loss (Carranza, & Newman, 1996).

Early onset periodontitis (EOP), also called juvenile periodontitis, includes advanced destructive lesions in children and adolescents (*Merck Manual*, 1998). The two major forms are prepubertal and juvenile periodontitis. Prepubertal periodontitis is associated with immunologic or other systemic problems such as Papillon-Lefevere syndrome, hypophosphatasia, agranulocytosis, Down's syndrome, leukocyte adhesion deficiency, neutropenias, Chediak-Higashi syndrome, AIDS, diabetes mellitus, cancer, histocytosis X and Ehlers-Danlos syndrome (type VIII). The signs and symptoms have an abrupt onset usually marked by malaise, acutely painful bleeding gingiva, hypersalivation, fetid breath odor and ulcerations noted on the gingiva and interdental papillae (Carranza, & Newman, 1996). The interdental papillae are usually characterized by a "punched-out" appearance, covered by a grayish membrane, and the gingiva bleed with the slightest pressure or irritation (*Merck Manual*, 1998).

Necrotizing ulcerative periodontitis (NUP) usually follows repeated long-term episodes of acute necrotizing ulcerative gingivitis and exhibits deep interdental osseous craters, which are usually localized (Carranza, & Newman, 1996). However, in advanced cases, the disease may spread to adjoining soft tissue with intraseptal sequestration of alveolar bone. The organisms implicated in the pathogenesis of these conditions include a mixture of aerobic and anaerobic gram-negative bacteria, spirochetes and yeast. In recent years, the incidence of NUP has increased among individuals who are HIV positive, malnourished, emotionally stressed, sleep/rest deprived, physically debilitated, smokers, homeless and individuals with poor oral hygiene. Often these clients present with regional lymphadenopathy and complaints of extreme pain during activities such as swallowing and talking. The management of clients with NUP generally involves debridement of necrotic tissue, irrigation with povidone-iodine/chlorhexidine oral solution and systemic antibiotic therapy (*Merck Manual*, 1998).

## RISK FACTORS

### Socioeconomic Risks

In the U.S., several research studies conducted over the past 30 years have identified specific risk factors in the development of periodontal diseases. These risk factors include: age, gender, race, education, income, place of residence and geographic location. A consistent finding in studies on the prevalence of periodontal diseases implicates age as a major risk factor. The progression and accumulation of the effects of periodontal disease are more severe among older adults (over age 65 years) than in any other age group. This does not imply that aging causes an increase in periodontal disease; no cause and effect relationship has been determined (Carranza, & Newman, 1996). However, a growing number of older Americans are experiencing chronic diseases and taking medications that affect periodontal health, which contributes to the overall problem (U.S. Preventive Services Task Force, 1996). Thus, it is not surprising that, approximately 44% of elderly adults no longer have their natural teeth as a result of periodontal diseases (www.cdcinfo@cdc.gov).

With regard to gender and race, males have a higher prevalence and severity of periodontal disease than females; and the prevalence of periodontal disease with tooth loss has been consistently higher among African-Americans than among Whites and Hispanics. Studies also indicate that the higher the educational level and the more professional the occupation, the less the incidence of periodontal disease. In addition, the prevalence of periodontal disease declined by 19% as the income of a family increased from less than $20,000 to $40,000 per year (Carranza, & Newman, 1996). Unfortunately, among low-income families and migrant workers, up to 80% have tooth and gum diseases that remain untreated and often result in pain, dysfunction and poor appearance (www.cdcinfo@cdc.gov)

According to Spradley (1991), the prevalence and severity of periodontal diseases are higher in rural areas than in urban areas. Children between the ages 6 and 11 years of age and youth between the ages of 12 to 17 living in the South have a slightly higher incidence of periodontal disease than those liv-

ıg in the Midwest and West. Otherwise, there have been no gnificant regional differences in the prevalence and severity f periodontal disease for adults in the U.S.

### ifestyle Risks

As documented in Beare and Myers (1994), several etiogic risk factors for periodontal disease have been identified. hese risk factors include: inadequate oral hygiene practices; utritional deficiencies in vitamins and minerals, i.e., A, B omplex, C, D, calcium, phosphorus and fluoride; pregnan-; trauma from bruxism; adverse habits such as smoking nd betel nut chewing; and the lack of professional dental re.

There is a strong positive association that exists between oor oral hygiene and periodontal disease in adults. Active eriodontal disease is rarely found in the absence of oral ebris (plaque) or calculus. Plaque and calculus are the priary etiologic factors for dental caries, resulting in a gradual athologic process of disintegration and dissolution of tooth ructures. The location and rate of plaque formation is irectly influenced by oral hygiene practices (toothbrushing, ossing and using fluoridated toothpaste or gel), diet (conuming foods high in refined carbohydrates) and salivary omposition and flow rate. Xerostomia, dry mouth associatd with diminished salivation, increases the risk for perioontal disease. The excessive deposition of plaque and calulus on teeth and in supragingival tissue sites contributes to ne proliferation of the infectious organism, *Streptococcus utans*, which causes tooth demineralization and the eventul development of periodontitis (Carranza, & Newman, 996).

Vitamin and mineral deficiencies greatly predispose indiiduals to developmental deficits and severe infectious and nflammatory changes in the tooth and periodontium. Of reat significance is the increase in the prevalence and incience of gingival or periodontal disease in individuals who ave low bone concentrations of fluoride, an inorganic ion nat essentially concentrates in teeth and bone, forming a rystal matrix that prevents acid erosion and demineralizaon. When fluoride is applied to a tooth surface in highly oncentrated forms (> 1 mg/day), as a gel or solution, it will eposit as calcium fluoride on or in the outer enamel and entin significantly reducing the incidence of tooth decay by 0% to 40% (Ekstrand, Fejerskov, & Silverstone, 1988).

Gingival changes in pregnancy are most noted during the ird trimester and are associated with the increased levels of rogesterone, which produce dilation and tortuosity of the ngival microvasculature, circulatory stasis and increased sceptibility to mechanical irritation. Any previously existg gingivitis or periodontal disease will be aggravated by the ormonal changes evident in pregnancy, along with creased tooth mobility, pocket depth and gingival fluid proaction (Woodall, 1993; Carranza, & Newman, 1996).

A recent study in the *Journal of the American Dental sociation* (reported online at www.ada.org/newsrel/9712/-05.html) reported that EOP sufferers who smoke have more severe periodontal disease and are at higher risk for separation of the gum from the tooth than those with the disease who do not smoke. Tobacco use increases irritation and inflammation of the gingival tissues and lowers resistance to infectious organisms (Ekstrand, Fejerskov, & Silverstone, 1988; Carranza, & Newman, 1996).

In another report, researchers from the University of Helsinki [Finland] and the University of Southern California dental schools reviewed recent studies that suggest two major periodontal bacteria are transmitted among family members: *Actinobacillus actinomycetemcomitants* and *Porphyromonas gingivalis*, causative agents in advanced periodontitis. These periodontal bacteria inhabit the mouth on the tongue, mucosal surfaces and in saliva and thrive within inflamed periodontal pockets. Researchers have recently suggested that the periodontal bacteria can spread via saliva from one person to another; a high incidence was noted in 20% to 30% of married couples (www.ada.org/newsrel/9712/nr-05.html). Additional oral pathology and associated causative factors in the development of periodontitis are listed in Table 1.

In advanced nursing practice, early identification and management are critical to preventing disease and complications. While all adult clients should be evaluated for periodontal disease, it is imperative that those individuals who are at high risk be identified for early and expedient intervention. As previously mentioned, age, race, gender, income and health status are critical factors in determining who is most at risk for periodontal disease and its complications.

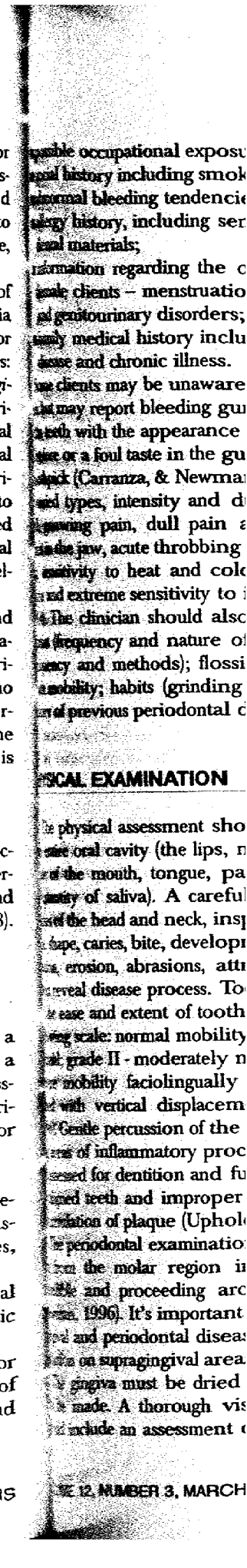
### Clinical Management

Proper assessment, diagnosis and planning are vital to effective management. The diagnosis of periodontal disease is determined after careful analysis of the history, clinical signs and symptoms and physical assessment (Uphold, & Graham, 1998).

## HISTORY

During the initial meeting, the clinician should perform a comprehensive health assessment. This should include a health history, including dental history, and physical assessment that is focused on the client's gingival and tooth integrity. Specifically, the health history for the client, with actual or potential periodontal disease, should include the following:

- client's current overall health status;
- history of all acute or chronic medical problems, especially, cardiovascular, hematologic, endocrine and gastrointestinal conditions, and any infectious diseases, including sexually transmitted diseases such as HIV;
- previous surgeries and hospitalizations, including oral surgeries and any untoward events such as anesthetic reactions, hemorrhagic or infectious complications;
- all medications being taken, whether prescribed or obtained over the counter and all possible effects of these medications on oral tissues, i.e., anticoagulants and corticosteroids;



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There is a strong positive association that exists between poor oral hygiene and periodontal disease in adults. Active periodontal disease is rarely found in the absence of oral debris (plaque) or calculus. Plaque and calculus are the primary etiologic factors for dental caries, resulting in a gradual pathologic process of disintegration and dissolution of tooth structures. The location and rate of plaque formation is directly influenced by oral hygiene practices (toothbrushing, flossing and using fluoridated toothpaste or gel), diet (consuming foods high in refined carbohydrates) and salivary composition and flow rate. Xerostomia, dry mouth associated with diminished salivation, increases the risk for periodontal disease. The excessive deposition of plaque and calculus on teeth and in supragingival tissue sites contributes to the proliferation of the infectious organism, *Streptococcus mutans*, which causes tooth demineralization and the eventual development of periodontitis (Carranza, & Newman, 1996).

Vitamin and mineral deficiencies greatly predispose individuals to developmental deficits and severe infectious and inflammatory changes in the tooth and periodontium. Of great significance is the increase in the prevalence and incidence of gingival or periodontal disease in individuals who have low bone concentrations of fluoride, an inorganic ion that essentially concentrates in teeth and bone, forming a crystal matrix that prevents acid erosion and demineralization. When fluoride is applied to a tooth surface in highly concentrated forms (> 1 mg/day), as a gel or solution, it will deposit as calcium fluoride on or in the outer enamel and dentin significantly reducing the incidence of tooth decay by 20% to 40% (Ekstrand, Fejerskov, & Silverstone, 1988).

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- all medications being taken, whether prescribed obtained over the counter and all possible effects these medications on oral tissues, i.e., anticoagulants a corticosteroids;

- possible occupational exposures;
- social history including smoking, alcohol or drug abuse;
- abnormal bleeding tendencies;
- allergy history, including sensitivity to foods, drugs and dental materials;
- information regarding the onset of puberty – and for female clients – menstruation, pregnancies, menopause and genitourinary disorders;
- family medical history including history of periodontal disease and chronic illness.

Some clients may be unaware of any dental or oral problems but may report bleeding gums, loose teeth or spreading of the teeth with the appearance of spaces where none existed before or a foul taste in the gums relieved by digging with a toothpick (Carranza, & Newman, 1996). There may be pain of varied types, intensity and duration, including constant, dull, gnawing pain, dull pain after eating, deep radiating pains in the jaw, acute throbbing pain, sensitivity when chewing, sensitivity to heat and cold, burning sensation in the gums and extreme sensitivity to inhaled air (Beare, & Myers, 1994). The clinician should also inquire about visits to the dentist (frequency and nature of treatment); tooth brushing (frequency and methods); flossing; orthodontic treatments; tooth mobility; habits (grinding or clenching of teeth); and history of previous periodontal diseases.

## PHYSICAL EXAMINATION

The physical assessment should include: examination of the entire oral cavity (the lips, mucous membranes, gingiva, floor of the mouth, tongue, palate, oropharynx and quality/quantity of saliva). A careful examination of the lymph nodes of the head and neck, inspection of the teeth for color, size, shape, caries, bite, developmental defects, abnormal formation, erosion, abrasions, attrition, staining and mobility often reveal disease process. Tooth mobility is graded based on the ease and extent of tooth movement according to the following scale: normal mobility; grade I - slightly more than normal; grade II - moderately more than normal; grade III - severe mobility faciolingually and/or mesiodistally, combined with vertical displacement (Carranza, & Newman, 1996). Gentle percussion of the teeth may elicit pain, indicating areas of inflammatory process. Finally, the teeth should be assessed for dentition and functional occlusion; irregularly aligned teeth and improper proximal contacts favor the accumulation of plaque (Uphold, & Graham, 1998).

The periodontal examination should be systematic, starting from the molar region in either the maxilla or the mandible and proceeding around the arch (Carranza, & Newman, 1996). It's important to detect the earliest signs of gingival and periodontal disease. Plaque and calculus accumulation on supragingival areas can be directly observed.

The gingiva must be dried before accurate observations can be made. A thorough visual examination the gingiva should include an assessment of color, size, contour, consistency, surface texture, position, ease of bleeding and pain (Carranza, & Newman, 1996). Gingival inflammation will be manifested as edema and/or fibrosis.

## CLINICAL MANAGEMENT

Clinical management of the client with periodontal disease will depend on the type and severity of the disease. For clients with SPP secondary to chronic gingivitis or systemic disease, prompt referral to a dentist is critical for the prevention of long-term complications. However, for SPP, secondary to drug-induced, fibrotic, gingival hyperplasia, 3 mg/day of folic acid with meticulous plaque removal is of benefit in reducing the incidence and severity of the adverse effect (Woodall, 1993). Rapid progressive periodontitis must be treated immediately and aggressively by dentists specializing in periodontics and periodontal surgery. Necrotizing ulcerative periodontitis is best treated by gentle, thorough, local debridement using warm, normal saline or 1.5% peroxide solution for 2-3 days, analgesia, adequate nutrition, increased fluid intake, and rest and oral hygiene using a soft bristle tooth brush. If fever or signs and symptoms of extension of infection are present, penicillin G or V, erthromycin or tetracycline should be prescribed (Beare, & Myers, 1994). All clients should be referred to a dentist for restoration of gingival health.

Measures aimed at the prevention of periodontal disease should ensure water fluoridation in all communities, as well as comprehensive self-care education and community-based screening programs. The incidence of dental caries and periodontal diseases has been decreased significantly by the fluoridation of community water supplies (U.S. Preventive Services Task Force, 1996). Fluoridation of water is the most inexpensive way to deliver the benefits of fluoride to all residents of a community. Even though the per capita cost of water fluoridation over an entire lifetime comes to less than the cost of one dental filling, more than 100 million American children and adults do not have access to water containing fluoride to protect their teeth (www.cdcinfo@cdc.gov). In fact, only 75% of the U.S. population is served by community-based fluoridated water systems, 62% of which actually have optimal levels of fluoridation (U.S. Preventive Services Task Force, 1996). Advanced practice nurses can play a pivotal role in convincing city and state politicians to invest tax dollars in developing community water systems that will provide adequately fluoridated water.

According to the American Dental Association, the prevention of periodontal disease involves educating clients to brush teeth thoroughly twice a day, using a soft bristled toothbrush with fluoridated toothpastes and mouth rinses. Daily flossing or the use of interdental cleaners is quite effective in removing bacteria and food particles from between teeth. Clients must be educated to eat foods high in protein, vitamins and minerals, and low in refined carbohydrates. Regular dental checkups and professional cleaning are essen-

tial in the prevention of periodontal diseases.

According to Caton, Ciancio, Crout, Hefti, and Polson (1998), research studies indicate the use of Periostat™ (CollagGenex Pharmaceuticals: Newtown, PA) is an effective antibiotic therapy in the reduction of gingivitis and periodontal disease in adults. Periostat™ is a systemically delivered collagenase inhibitor available as a 20 mg capsule of doxycycline hyclate for oral administration. Periostat™, administered BID reduces the elevated level of collagenase activity in the gingiva of adults with periodonitis. However, Periostat™ has not been shown to be a substitute for meticulous home care, routine cleanings and appropriate treatments designed to minimize bacterial load. Each practitioner must collaborate with dental health professionals to determine the usefulness of Periostat™ in light of the available data and the needs of the individual patient.

The clinician must provide community leadership in building coalitions, developing partnerships and constituencies with community officials, health care professionals and other leaders in order to develop programs in schools, consumer-based industries, churches, health departments and community centers to promote dental health education, routine screening of children and adults, and funding to provide free dental health services. It's imperative that individuals at risk for periodontal disease are identified early and that effective clinical interventions are provided to prevent the serious complications of periodontal disease.

In conclusion, periodontal diseases are, in many cases, preventable with the appropriate dental health practices. Advanced practice nurses must be knowledgeable of the causative agents, risk factors and pathophysiology of periodontal disease. There are many implications for advanced practice nurses, regardless of the setting, to initiate early identification, prevention and management of clients with actual or potential periodontal diseases.

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